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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE
DISEASES OF INFANTS AND CHILDREN
JOHN FITCH LANDON, M.D., Editor

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THE POISON CAUSE OF POLIOMYELITIS AND OBSTRUCTIONS TO ITS INVESTIGATION*

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The disease that we now know as poliomyelitis was not designated as such until about the middle of the 19th Century. Prior to that, it was designated by many different names at various times and in different localities.[1, 2] The simple designations, paralysis, palsy and apoplexy, were some of the earliest names applied to what is now called poliomyelitis.

Paralysis, resulting from poisoning, has probably been known since the time of Hippocrates (460-437 B.C.). Boerhaave,[3] Germany, (1765) stated: "We frequently find persons rendered paralytic by exposing themselves imprudently to quicksilver, dispersed into vapors by the fire, as gilders, chemists, miners, etc., and perhaps there are other poisons, which may produce the same disease, even externally applied." In 1824, Cooke,[4] England, stated: "Among the exciting causes of the partial palsies we may reckon the poison of certain mineral substances, particularly of quick silver, arsenic, and lead. The fumes of these metals or the receptance of them in solution into the stomach, have often caused paralysis."

Colton[5] (1850) mentions the case of a patient who swallowed some arsenic accidently and was admitted to the hospital. The primary effects of the poison had been successfully combatted with proper remedies, but seven days afterward he became paralyzed. It is significant to note that there was a latent period of several days before the paralysis appeared since this delayed reaction is comparable to the incubation period in infectious diseases.

Vulpian[6] (1879) experimentally produced paralysis of the extensor muscles of a dog by lead poisoning. The lesions, consisting in colloid degeneration and cell atrophy of the anterior horn cells of the spinal cord were pronounced by Vulpian as poliomyelitis. Adamkiewitz[7] (1879) reported two parallel cases, one of poliomyelitis and one of lead poisoning.

In 1881, Popow[8] of St. Petersburg, published an essay upon the pathological anatomy of arsenical paralysis as produced artificially in animals. The work of Popow was carried out under the guid-

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ance of the distinguished neurologist and microscopist, Professor Mierzeyeski. Popow concluded that arsenic, even in a few hours after its ingestion, may cause acute central myelitis or acute poliomyelitis.

During an epidemic of poliomyelitis in Australia in 1897, Altman[9] pointed out that phosphorus had been widely used by farmers for fertilizing that year. This observation may be of significance since in recent years organic phosphorus insecticides, such as parathion, have been suspected as possible causes of poliomyelitis.

Onuff[10] (1900) reported a case of a painter with flaccid paralysis of both legs, in whom the autopsy showed lesions characteristic of poliomyelitis.

Obrastoff[11] (1902) reported a case of acute poliomyelitis resulting from arsenic poisoning. Phillippe and Gauthard[12] (1903) reported a case of anterior poliomyelitis from lead poisoning.

Gossage[13] (1902), writing on infantile paralysis, says: "The nerve cells or fiber may be acutely disabled by the action of some poison circulating in the blood, and it is possible that such poison would only temporarily impair their functions or so seriously affect them that recovery would be impossible."

Dr. David E. Edsall[14] (1907), writing on the pathology of carbon monoxide poisoning in Osler's System of Medicine, states: "Peripheral neuritis had repeatedly been described and poliomyelitis and disseminated encephalitis have been seen."

Collins and Martland[15] (1908) reported a case of poliomyelitis in a man, 38 years of age, which resulted from the use of potassium cyanide as a silver polish. The illness began with diarrhea, followed by headache and pain and stiffness in the back of the neck. About eight days after the onset of the illness, he became paralyzed. In discussing Collins and Martland's paper, Larkin stated that he had seen one instance of this disease following potassium cyanide poisoning.

Collins and Martland poisoned several rabbits with potassium cyanide and found pathological lesions in the spinal cord similar to those found in cases of poliomyelitis.

In the spring of 1930, there occurred in Ohio, Kentucky, Alabama, Mississippi and other states an epidemic of paralysis.[16, 17] The patients gave a history of drinking commercial extract of ginger. It is estimated that at the height of the epidemic there

were about 500 cases in the Cincinnati district alone. The cause of the paralysis was subsequently shown to be triorthocresyl phosphate in a spurious Jamaica ginger. Death resulted not infrequently from respiratory paralysis similar to the bulbar paralysis deaths in poliomyelitis. On pathological examination, the anterior horn cells of the spinal cord in these cases showed lesions similar to those of poliomyelitis.

These incidents show that epidemics of poisoning occur and, furthermore, that epidemic diseases do not always indicate that they are caused by infectious agents. Moreover, following the ingestion of the spurious Jamaica ginger, the symptoms appeared two to ten days later. In some cases a longer time elapsed. This latent period is comparable to the incubation period of infectious diseases. As a matter of fact, the incubation period of poliomyelitis is commonly stated to be seven to 10 days on the average with considerable variation in either direction. The so-called incubation period in poliomyelitis and the latent period in these cases of poisoning, therefore, are strikingly similar in length.

Leenhardt et al.[18] (1951) described acrodynia in the course of three cases of acute poliomyelitis. Some authorities have considered acrodynia to be caused by a poison. Elmore[19] (1948) reported two cases of this disease following the ingestion of mercury and Warkany and Hubbard[20] (1951) found mercury in the urines of 38 (92.7 per cent) of 40 acrodynia patients. Meyerhofer[21] (1939) reported that infantile acrodynia may immediately follow certain forms of atypical poliomyelitis, especially encephalomyelitis. Mercury is used as an insecticide and a fungicide and the above clinical observations indicate that it might be a factor in producing some cases of poliomyelitis.

Gougerot[22] (1935) reported that during arsenical therapy for syphilis, poliomyelitis developed in two patients, and lethargic encephalitis followed by Parkinson's disease in one.

In 1936, during a campaign to eliminate yaws in Western Samoa by the injection of arsenicals, an epidemic of poliomyelitis appeared simultaneously.[23] In one community all of the patients developed paralysis in the same lower limbs and buttocks in which they had received the injections and this pattern was repeated in 37 other villages, whereas there was no paralysis in uninoculated districts. The natives accused the injections as the cause of the

epidemic of poliomyelitis. Most of the cases of paralysis occurred one to two weeks after the injection of the arsenic.

The foregoing reports indicate that poisons can cause poliomyelitis. It would appear that not any one poison in particular would be responsible for all cases of poliomyelitis but the effect of any one of several could produce the same ultimate result. When a disease is known to be caused by a poison, it is obvious that a search for a germ or virus in relation to it would not be made. Conversely, if a so-called virus is believed to be associated with the disease, then the possibility of poisoning as the cause of the disease would not be considered. It will be shown, moreover, that some so-called virus diseases and virus inclusions can be caused by poisons.

Dr. Robert W. Lovett[24] of the Massachusetts State Board of Health (1908), describing the epidemic of poliomyelitis in Massachusetts in 1907, and after reviewing the medical literature on experimental poliomyelitis, states: "The injection experiments prove that certain metallic poisons, bacteria and toxins have a selective action on the motor cells of the anterior cornua when present in the general circulation; that the paralysis of this type may be largely unilateral; that the posterior limbs are always more affected than the anterior; and that the lesions in the cord in such cases do not differ from those in anterior poliomyelitis." It appears to be of great importance that the various poisons, lead, arsenic, mercury, cyanide, etc., found capable of causing paralysis are employed in relation to articles of food that are used for human consumption.

There are two abnormal findings in cases of poliomyelitis that point strongly to poisoning as the cause of this disease. One consists in the appearance of increased amounts of porphyrin in the urine[25]; the other is the presence of increased amounts of guanidine in the blood. It is a well-known fact that porphyria can follow poisoning by a number of chemicals. Guanidine[26, 27] has been found in increased amounts in the blood in arsenic, chloroform, and carbon tetrachloride poisonings.

The fact that ascorbic acid has been effective in the treatment of poliomyelitis[28-31] appears justly to imply that this disease has a poison cause. Ascorbic acid has been used as a reducing agent in the treatment of poisoning resulting from a number of toxic

agents, including coal tar antipyretics, nitro compounds, analine, cyanide, benzene, lead, arsenic, etc.[32-40] Paralleling these modern scientific investigations is the observation over a century ago that lime juice and lemon juice were protective against the poisoning by fish which sometimes resulted in paralysis.[41, 42] This early observation is perhaps the principle reason why lemon juice is customarily served today when fish are eaten.

The fact that methylene blue,[43] another reducing agent, is effective in the treatment of poliomyelitis also points to the poison cause of this disease. Methylene blue has been used as an antidote in the treatment of nitrite, cyanide, carbon monoxide and other poisonings.

Another fact that strongly implies that human poliomyelitis is caused by a poison is found in the recent report (1951) by Dr. Irwin S. Eskwith[44] of Bridgeport, Conn., that BAL (dimercaprol) was effective in bringing about complete recovery in a moribund 4½-year-old girl with bulbar poliomyelitis. BAL counteracts the effects of poisons; it has been shown not to be effective in infectious diseases.

RELATIONSHIP OF HARVEST TO POLIOMYELITIS

It is a well-known fact that certain plants have natural poisonous properties, for example, toadstools, but it is not generally realized that some natural foods that are usually considered to be wholesome and nutritious may at times be poisonous. Such factors as drought, or excessive rainfall, fertilization with nitrates, and the character of the soil may result in the development of poisons in plants that are ordinarily edible. The frequent observation that epidemics of poliomyelitis often occur and not infrequently are confined to districts affected by drought is noteworthy in this connection.

The finger of suspicion has for centuries been pointed toward food as the cause of paralytic affections occurring during the season when poliomyelitis epidemics are known to occur.

Creighton[45] (1891), writing on epidemics in Britain, stated that in 1383, and before that there is unmistakable reference to many fatalities, as well as serious maladies caused by eating damaged fruit.

In the 16th Century epidemics in which no symptom or sign of polioencephalitis, poliomyelitis, or cerebrospinal meningitis was

lacking, ergot, bad bread, sour apples, mushrooms and the like were mentioned as causes.[46]

Cholera morbus, a disease which was known to physicians in the past and in some respects resembles the poliomyelitis of the present, was first described by Sydenham[47] in 1669. The cause of this disease was attributed by medical writers since the time of Sydenham to unripe fruits and vegetables, fish, shellfish, water, miasm, etc. Cruvelier[48] (1833), as well as others, pointed out that the patient presented the symptoms of poisoning, a fact which he says was noted by Morgagni, and almost every writer since his time.

Benjamin Rush[49] (1799), in describing summer and autumnal fevers, points out the effects of drought on leaves, vegetables and fruits, denoting the inferior size and quality of the latter. He observed illnesses among birds and beasts and pointed out that the brain was not infrequently affected in children. He advised against the use of unripe and decayed fruits at such times.

The failure of the principle food crop—the potato—in Ireland, in 1817 and 1849,[50, 51] caused the people to eat new potatoes dug soon after the seed was planted as well as weeds and shellfish. Food was eaten raw or improperly cooked because of lack of fuel. Epidemic fever, among other names, was the designation for the illnesses that resulted. Paralytic manifestations were not infrequently observed as sequelae. Contemporary physicians noted that the effect in some cases resembled that of a concentrated poison.

Harris[52] (1920) pointed out that potato poisoning from new and sprouting potatoes can result in vomiting, diarrhea, colicky pains, headache, slight fever and prostration. Partial paralysis may at times occur. Colton[53] (1917) described an epidemic of poliomyelitis which he ascribed to the eating of potatoes that had been affected by drought.

In 1907, Dr. H. C. Emerson,[54] Massachusetts State Inspector of Health, District 14, investigating an epidemic of poliomyelitis in that state, made a careful inquiry regarding the diet. No infant who was fed exclusively on the breast developed poliomyelitis. He found in six cases that fruit and berries had been a large item of the diet. In the cases of two infants, bananas and berries had been given in the diet in addition to breast milk. In three cases of poliomyelitis, the illness was attributed to the eating of large amounts of blackberries and blueberries. In one case the illness

was credited to eating heartily of English mulberries. In 39 instances it was stated that food supplies were bought from fruit and vegetable peddlers in their localities.

Milk, which at times is known to contain toxins resulting from poisonous plants eaten by cows, especially when overgrazing because of drought, can cause illness in humans. Dingman[55] (1916) reported a milk-borne epidemic of poliomyelitis and several similar outbreaks have been reported since then that were traceable to milk. Dr. Marsh Pitzman[56] (1928) insisted that poliomyelitis is caused by poisonous alkaloids eaten by cows, the poison being conveyed to humans through milk. Louis Bromfield,[57] in his book, "Out of the Earth," endorses this explanation of poliomyelitis.

Chapman,[58] raised the question of food poisoning to explain the epidemic of poliomyelitis in England in 1947, when he stated: "Is it not possible that the present prevalence of infantile paralysis may, in part at any rate, be due to some article in our restricted and modified dietary? In the Central Provinces of India there were after famine years widespread outbreaks of a form of spastic paralysis, caused by the consumption of the millet, Lathyrus sativa. In ordinary times this was used only as an adjunct to the other grains, and it was only when used as a staple diet that the paralysis occurred. Is it not possible that in the modification of the austerity diet we may have introduced some article which may be causing this widespread epidemic?"

Toomey and August[59] (1932) pointed out that some authors thought that poliomyelitis is a disease of gastrointestinal origin which might follow the ingestion of foodstuffs. In 1933,[60] they noted that the epidemic peak of poliomyelitis corresponds with the harvest peak of perishable fruits and vegetables. They called attention to the fact that the disease occurs only in those countries which raise the same type of agricultural products. Dr. C. W. Burhans,[60] one of the colleagues of the authors, thought that green apples might be a factor in the etiology of poliomyelitis. Toomey et al.[61] (1943) point out that there is frequently a history of dietary indiscretions previous to an attack of poliomyelitis. They suspected that a virus could be found on or in unwashed fruit or in well water during epidemics of poliomyelitis. Every year for eight years, therefore, grapes, apples, peaches, and pears were collected from the vineyards and trees in Northern Ohio at the time of the

ripening. In none of their studies was the so-called virus of poliomyelitis demonstrated when the washings of the fruit or the well water were injected into experimental animals. However, no chemical tests were made to determine whether or not a chemical substance on or within the fruit or in the well water, acting by oral ingestion to produce poliomyelitis, was present.

Draper[62] (1935) recorded a series of cases of poliomyelitis which he postulated originated from a Greek fruiterer. All of the cases were in contact with the Greek as business associates, relatives or customers, and there was nothing in the evidence to point to infection being carried by the Greek himself other than the fruit he supplied.

Leavell[63] (1936) suggested that the ingestion of uncooked fruits and vegetables might be responsible for poliomyelitis.

Barber[64] (1939) reported four cases of poliomyelitis that developed simultaneously on the same day from the eating of strawberries in a single house of a boarding school. He says that the simultaneous onset of these cases resembled food poisoning. The seasonal and climatic incidence of poliomyelitis, he points out, agree closely with the seasonal increase in the consumption of fresh garden production. He says that the epidemiological distribution of poliomyelitis resembles food poisoning.

Chenault[65] (1941) noted that the history of poliomyelitis points to a "suggested parallelism between a number of epidemics and the appearance of fresh fruits and vegetables."

Goldstein et al.[66] (1946) reported an epidemic of polioencephalitis at a naval training school among the cadets. The epidemic was explosive in character and involved over 100 persons. Epidemiological evidence suggested that some food served in the mess hall was the cause of the disease.

Kovar[67] (1945) noted the frequency of acute gastrointestinal upsets at the time of epidemics of poliomyelitis in Nebraska in the years when severe drought and economic depression were prevalent. The symptoms and signs of many of his cases were similar to those of preparalytic poliomyelitis, but the patients had negative spinal fluids and recovered without paralysis. One case, a boy aged 13 years, had a diet for a few days prior to his illness which consisted of foods of harvest that are capable of producing gastrointestinal upsets. In another case, a boy 9 years of age, had eaten a

number of apples along with other fresh harvest foods the day before the onset of his illness. A third child, age 15 years, had been drinking large quantities of water and had had a very unbalanced diet for a week prior to his illness. Kovar considered the symptoms in these cases that he had observed as gastrointestinal in origin because the diets in each of his cases consisted in foods capable in themselves of producing gastrointestinal upsets. He says: "Because of the economic depression, proper food could not be purchased, and because of the drought proper food could not be obtained or raised."

Gebhardt and McKay[68] (1946) found during an epidemic of poliomyelitis in Utah that of a total of 206 patients surveyed, 192 persons, or 93.2 per cent, had one to two weeks prior to the onset of the disease eaten fresh fruits. The authors found in Utah, New York and California, during 1943, that the cases of poliomyelitis paralleled the harvest peaks. Most of the multiple cases in families were found to have developed at the same time, suggesting means other than contact as the mode of spread. Among the fruits more commonly eaten were apples, peaches and pears; tomatoes headed the list of vegetables. The authors stated that the data appeared to fit into the jigsaw puzzle of epidemic poliomyelitis.

Scobey[69] (1946) in an article entitled, "Food Poisoning as the Etiological Factor in Poliomyelitis" pointed out that the frequent reports of poliomyelitis following the ingestion of mushrooms, fruits, vegetables, and milk could be attributed to the cyanide content of these foodstuffs. In a later report,[70] he pointed out that water, under certain conditions, especially drought, could also contain cyanide.

It is well-known to veterinarians and farmers that certain grasses, including Johnson grass, Sudan grass, millet, etc., affected by drought, fertilization by nitrates, and other factors, contain a high percentage of cyanophore glucosides capable of producing hydrocyanic acid resulting in paralysis and death by respiratory paralysis of cattle. Leaflet 88, U. S. Department of Agriculture, entitled, "Poisoning of Livestock by Plants that Produce Hydrocyanic Acid" describes the effects of these plants on cattle. Cyanide is found also in plants that belong to the rose family, which includes many of our common fruits, and in members of the mustard family, including cabbage, etc. It has been demonstrated that mem-

bers of the mustard family have goitrogenic properties in addition to the effects that have been mentioned.

Abbott[71] (1948), of Auckland, New Zealand, stated: "The public has always been fully convinced that they caught poliomyelitis from one another by direct infection. The 'germ' idea is indeed deeply ingrained in both the profession and the public. It will be many years before our prolific writers of medical textbooks attain the degree of sophistication that would enable them to understand how and why poliomyelitis would be much more likely to be contacted from the flour-bag, or some other homely article of food, rather than from their neighbors."

Adamson et al.[72] (1949) reported an epidemic of poliomyelitis in the Arctic involving 78 Eskimos between the ages of 8 and 64 years. The explosive and devastating character of the epidemic strongly suggests food poisoning as the etiological factor. This conclusion is borne out by noting that there had been a scarcity of food and there was gouging of food when a supply was obtainable; the Eskimos had little regard for the state of preservation of the food and showed marked preference for meat when it was "high"; the eating habits were irregular, with a minimum amount of preparation and cooking; not a child under three years of age was afflicted with the disease since Eskimo mothers nurse their babies until that age; gastrointestinal disorders were noted to be more prevalent than usual during epidemics of poliomyelitis.

Barondes[73] (1949) points out that a study of the epidemiology of poliomyelitis shows a definite correlation with the harvesting of fruit and vegetable crops and to changes in climate, weather and humidity. The harvesting of such fruits as cherries, grapes, berries, apricots, etc. and the edible vegetables, as lettuce, radish, cucumbers, etc. usually from June to September, corresponds with the period of poliomyelitis epidemics, Barondes points out.

Toomey et al.[74] (1949) made some important experimental observations that appear to show a correlation of the poison and virus theories of poliomyelitis. They considered it possible that a food (fruit) which enters the gastrointestinal tract could in some way act as a precursor or catalytic enzyme on a normal constituent of the tract and accelerate the production of poliomyelitis. Various materials, together with fruit extracts, were tested.

When supernates of peach skin mash were injected intracere-

brally into cotton rats, followed at intervals with intracerebral injections of the so-called poliomyelitis virus, accelerated production of paralysis occurred. Because of the presence of cyanophore glucosides in peach skins, a synthetic preparation, succinonitrile, was injected intracerebrally into the experimental animals. This chemical accelerated the production of the disease similar to that produced with the peach supernates. Toomey et al. emphasize that the injections of fruit supernates were made in a manner that does not occur under natural circumstances.

Sabin[75] (1951), although insisting on the virus etiology of poliomyelitis, implicates food and drink as important factors in the cause of this disease. He points out that measures which are often advocated to combat poliomyelitis epidemics are *not* warranted, such as (a) avoidance of crowds, large gatherings or sports events, (b) exclusion of children under 16 years of age from movies, churches, or schools, and (c) exclusion of poliomyelitis patients and suspects from general hospital wards.

The material presented above appears clearly to indicate that there is a correlation between the harvesting of fruits and vegetables and epidemics of poliomyelitis. Epidemics of this disease usually appear in June and reach their peak in August and September. They usually terminate with the first frost which kills vegetation. They are intensified in years of drought and are more prevalent in drought affected areas. The majority of poliomyelitis cases occur in rural areas and villages rather than in large cities. The foregoing material shows also that when careful inquiries are made concerning the diets of poliomyelitis patients prior to the onset of their illnesses, valuable information is obtained. Dietary histories, unfortunately, are rarely obtained from poliomyelitis patients because it is generally assumed that a virus derived from an active case or carrier is the cause of the disease. The implications should be obvious that investigation of foods eaten by the poliomyelitis victim prior to his or her illness should be carefully considered.

THE PRODUCTION OF SO-CALLED VIRUS DISEASES AND "VIRUS" INCLUSIONS BY POISONS

The public, as well as many physicians, is under the impression that viruses are living organisms comparable to a germ that enters the human, animal or plant to cause the disease. The scientists,

who are authorities on virus diseases, are in disagreement as to the nature of a virus.

It is not generally realized that some so-called virus diseases may result from the effects of poisons on the human body. Thus, herpes zoster may follow exposure to carbon monoxide or the administration of arsenic, bismuth, lipiodol, gold, mercury, tuberculin, alcohol, etc. An epidemic of herpes zoster and peripheral neuritis, similar to the "jake" paralysis epidemic in this country, followed the ingestion of arsenic in beer in Manchester, England in 1900.[76-78] The toxic agent was determined to be arsenic arising from dextrose made from starch by the use of crude sulfuric acid containing this poisonous substance.

Herpes simplex, another so-called virus disease, has followed the ingestion of alcohol, benzol, arsenobenzol, mercury, and the inhalation of ether, among other poisons. Van Rooyen[79] noted its appearance after sulfapyridine therapy. Herpes simplex has followed the injection of vaccines, milk and colloidal metals.

Inclusion bodies have been defined as products of virus activity or the elementary virus bodies themselves. Inclusion bodies have been found in poisoned humans and experimental animals.

Dalldorf and Williams[80] (1945) found large acidophilic inclusion bodies in the kidneys of rats poisoned by lead. Blackman[81] (1936) found intranuclear inclusion bodies in the tubular epithelium of the kidney and in the liver cells of 21 children dying from the effects of acute lead poisoning and lead encephalitis.

Cox and Olitsky[82] (1934) found that the injection into animals of aluminum hydroxide produced inclusion bodies similar to those seen in infectious encephalitis.

Van Rooyen and Rhodes,[83] in their textbook (1948), "Virus Diseases in Man," state: "Histological changes similar to those seen in infectious encephalitis may be produced by carbon monoxide poisoning, brain injury, arteriosclerosis, uremia, pregnancy toxemia and toxic agents like alcohol and lead."

Olitsky and Harford[84] (1937) were able to produce inclusion bodies indistinguishable from those observed in virus infections by the injections of aluminum compounds, ferric hydroxide and carbon.

MISTAKES THAT HAVE BEEN MADE IN THE PAST

Several commissions, appointed during the first quarter of this

century to investigate the cause of pellagra, concluded from their studies that pellagra was an infectious, contagious disease. Harris[85] (1913) was able to inject Berkefeld filtered tissue material from pellagra victims into monkeys to cause a corresponding disease in these experimental animals. He concluded from these experiments that a virus was present in the injected material and that it was the cause of pellagra. If the work of Harris had been followed exclusively, various strains of this "virus" might have been discovered and a vaccine, effective in experimental animals, might have been developed, as in the case of poliomyelitis. Today, as a result of unlimited research, however, we know conclusively that pellagra is not caused by a virus but rather that it is a vitamin deficiency disease. It is obvious that if the investigations of pellagra had been restricted to the virus theory, it would still be a mystery.

Years ago, the disease known as milksickness in humans and trembles in cattle was classified in medical textbooks as an infectious disease. Stevens[86] (1922) classified milksickness as a disease caused by a filterable virus in his textbook "The Practice of Medicine." This disease was fatal to Nancy Hanks, mother of Abraham Lincoln.

Milksickness appeared in epidemics, similar to poliomyelitis, beginning in June and terminating with the first frost. The disease had an incubation period similar to infectious diseases; there was often a history of exposure to other cases of the disease; and it had the power of self propagation similar to virus infections. It was well-known, as in the case of poliomyelitis, that chilling and fatigue would precipitate an attack of the disease. Like poliomyelitis, milksickness occurred more frequently in rural districts than in cities.

Farmers had suspected for many years that milksickness and trembles were caused by a poisonous plant eaten by cattle. In fact, Dr. Daniel Drake,[87] a general practitioner (1810), published an article entitled, "An Account of the Diseased State produced by taking into the Stomach the Milk or Flesh of Cows which has acquired deleterious properties from a vegetable sometimes eaten by these Animals." However, the insistence by bacteriologists in later years, who were able to influence the thinking of well-known clinicians, that these diseases were caused by a germ or virus pre-

cluded for some time serious consideration of the poison cause of these diseases.

About 30 years ago, it was shown conclusively that milksickness in man and trembles in cattle are caused by the poison tremetol, an oily alcohol. This poison occurs in white snakeroot and rayless goldenrod. When these plants are eaten by cattle, the poison is conveyed to humans through the milk, milk products or the flesh of the animals. Water borne epidemics have been reported which resulted from the poisoning of the water by these plants.

The discovery of tremetol as the cause of these diseases explained why epidemics began in June and terminated with the first frost*; why chilling and fatigue** were factors in precipitating an attack; and why they appeared to be contagious. Huffman and Couch[88] (1942) state regarding these diseases: "The cause of the disease was obscure and a great variety of guesses were current among both laymen and the medical profession. . . . The isolation of tremetol and the production of trembles by feeding poisonous butter to sheep completed the chain of evidence linking the plants to human milksickness and solved a mystery which for over 150 years had baffled physicians and terrorized dwellers in the rural districts."

The symptoms of milksickness in man resemble those of influenza or grippe, gastritis, and so-called ptomaine poisoning. As a matter of fact, so-called summer grippe or flu often occurs during epidemics of poliomyelitis. There were 10,000 cases in Cincinnati in 1947,[89, 90] which were thought to be related to poliomyelitis and were considered, therefore, virus infections. However, Matson[91] (1950), writing about poisonous plants, says that some physicians have expressed the opinion that mysterious outbreaks of so-called summer flu in the late summer are often due to milk contaminated with tremetol.

The observation that human and bovine outbreaks of tremetol poisoning occur simultaneously corresponds with similar observa-

*Although epidemics of milksickness and trembles usually occurred during the growth period of the poisonous plants, they appeared occasionally even during the winter because of the presence of the poison in stored hay, beef, pork, butter, cheese, honey, etc.

**It was the custom of people in affected districts to chase their beef cattle around with dogs until they were heated by the exercise; then if the cattle did not show any signs of the disease they were killed and eaten as beef without fear. Dixon[92] (1833) states concerning milksickness in man: "It is one of the peculiarities of the disease that the poison occasionally lies long dormant in the system, and only becomes active by some indiscretion or violent bodily exertion."

tions made during epidemics of poliomyelitis. Medical reports have shown repeatedly that paralytic diseases in horses, pigs, dogs, cats, ducks, chickens, etc. occur simultaneously in districts where epidemics of poliomyelitis are prevalent.

FACTORS PRECLUDING INVESTIGATION OF THE POISON CAUSE OF POLIOMYELITIS

It is obvious that in the study of poliomyelitis every possible cause, including the possibility of poisoning, should be investigated.

Since 1908—for 44 years—poliomyelitis research has been predominantly directed along only one line of investigation, i.e., the infectious theory. This single line of study, precluding other possibilities, including the poison cause of the disease, has resulted from two factors, (1) The Public Health Law,[93] and (2) the insistence, based entirely on animal experiments, that poliomyelitis is caused by a virus.

1. *The Public Health Law.* The inclusion of poliomyelitis in the Public Health Law as a communicable, infectious disease dates back to the early part of the 20th Century. At that time many diseases, now known to be neither communicable nor infectious, were considered to be caused by an infectious agent simply because they occurred in epidemics. The general attitude of that period is expressed by Sachs[94] (1911) in his statement: "In general, the epidemic occurrence of any disease is sufficient to prove its infectious or contagious character." The vitamin deficiency diseases, beriberi and pellagra, are outstanding examples of epidemic diseases that were formerly considered to be infectious and communicable according to the logic employed by Sachs. In fact, we find pellagra incorporated into the Public Health Law as a communicable disease in the State of Pennsylvania in the following rule and regulation adopted January 5, 1910: "That all physicians practicing within the limits of the state shall make immediate report of each and every case of uncinariasis duodenalis (hookworm disease) and pellagra and anterior poliomyelitis (infantile paralysis) occurring in their practice in the same manner that other communicable diseases are now by law and by rule and regulation of the State Department of Health reported to the health authorities." A State Health Officer[95] recently wrote to me as follows: "I think all of us will agree with you that in the past, as is still probably true,

public health rules and regulations and sometimes even public health laws, were influenced too much by what we did not know rather than by what we did know. This was probably an acceptable line of reasoning in the past, but with increasing public health education and greater understanding and cooperation from citizens, this justification becomes less acceptable."

The fact that an extensive epidemic of poliomyelitis was prevailing in the states of New York and Massachusetts in 1907, aroused the suspicion that the disease was infectious and communicable; it was therefore incorporated into the Public Health Law as such. However, conclusive evidence of contagiousness was not established during that epidemic nor in subsequent ones. Moreover, conclusive evidence of contagiousness was not established in 1949, during the greatest epidemic of poliomyelitis in recorded history, as shown by the records of the U. S. Public Health Service and the New York State Department of Health. Time Magazine,[98] commenting on these surveys, points out how, when and where people catch polio remained a mystery. In addition to the failure to prove contagiousness of human poliomyelitis, it has likewise been impossible to prove contagiousness of poliomyelitis in experimental animals. This fact will be considered in detail later.

As a result of the inclusion of poliomyelitis in the Public Health Law as a contagious, communicable or infectious disease, investigations regarding it are almost exclusively in the hands of specialists in virology and public health. The country doctor, general practitioner, and clinician have little or no opportunity to participate in poliomyelitis research under these circumstances. Yet, Dr. W. Ritchie Russell[97] of the Department of Neurology, United Oxford Hospitals, Oxford, England stated in 1950: "Clinical research into this disease is so much neglected that there are exciting discoveries waiting for anyone with time to give to this type of investigation."

Medical advances of the utmost importance have been made in the past by general practitioners. An outstanding example is the work of Dr. Edward Jenner, a general practitioner in Gloucestershire, England, whose observations and deductions brought about vaccination against smallpox. All advances in medicine do not result from laboratory experiments. Any doctor in any community, however small, and however limited his opportunities, may make a fundamental discovery, but he must be given the

opportunity to participate in the program and his observations and deductions must be given adequate consideration.

2. *Virus Research.* The more or less general acceptance of the idea that poliomyelitis is caused by a virus arose from experimental animal studies by Landsteiner[98] (1908) in Austria, and Flexner and Lewis[99] (1909) in the United States. These experiments showed that a substance obtained from poliomyelitis victims could produce a paralytic disease when administered to experimental animals. It has been assumed, as a result of these experiments that an exogenous virus is the cause of human poliomyelitis. Dr. Harold L. Amoss[100] stated in 1928: "By reason of the parallelism of the human and experimental disease it is believed that *inferences drawn from experiments with monkeys may be accepted with a certain degree of safety as applicable to the solution of problems in connection with human cases.*" The portal of entry of the so-called virus of poliomyelitis into the human body never has been established. The question of the portal of entry is summed up in the published reports of the International Poliomyelitis Congress[101] that was held in New York City in 1948. The Modulator stated: "We do not know too much about the portal of entry in human beings," and Dr. John R. Paul, of Yale University, stated: "I would say we do not know the portal of entry in human beings."

It was mentioned in the foregoing that human poliomyelitis has not been shown conclusively to be a contagious disease. Neither has the experimental animal disease, produced by the so-called poliomyelitis virus, been shown to be communicable. Rosenau[102] (1921) stated: "Monkeys have so far never been known to contract the disease 'spontaneously' even though they are kept in intimate association with infected monkeys." Twenty years later (1941), Dr. John A. Toomey,[103] a poliomyelitis authority, stated: "No animal gets the disease from another no matter how intimately exposed."

It is extremely difficult to understand how a human can contract poliomyelitis from another individual through dissemination of a virus by contact, carriers, excrement, unclean hands, unwashed fruits and vegetables, flies, etc. when a healthy animal in the same cage with an "infected" animal, exposed to all of these natural factors, remains unaffected. It appears obvious, therefore, that communicability should have been established conclusively both in humans and in experimental animals before poliomyelitis was in-

corporated into the Public Health Law as a communicable disease.

Some investigators have as a matter of fact pointed out that human poliomyelitis and the disease produced in experimental animals from human material, etc. are not the same disease. Toomey[104] (1935), for example, stated that intranasal and intracerebral inoculation of poliomyelitis virus in the monkey does not produce the same disease that is seen in man. Dr. Claus W. Jungeblut,[105] a well-known bacteriologist who has worked on the poliomyelitis problem for many years, recently stated (1950): "Whatever the final answer may be, it seems a reasonable statement at this time that the highly specialized, neurotropically fixed virus, which has been maintained in the past by intracerebral passage in rhesus monkeys, is more likely a *laboratory artefact* than the agent which causes the *natural disease* in man." The logical conclusion appears to be, therefore, that the laboratory experiments with the so-called virus of poliomyelitis are merely of academic interest and have no practical application to human poliomyelitis. Jelliffe,[106] 32 years ago, cautioned: "The purity of laboratory experiments is rarely repeated in nature."

For almost half a century poliomyelitis investigations have been directed towards a supposed exogenous virus that enters the human body to cause the disease. The manner in which the Public Health Law is now stated imposes only this type of investigation. No intensive studies have been made, on the other hand, to determine whether or not the so-called virus of poliomyelitis is an autochthonous chemical substance that does not enter the human body at all, but simply results from an exogenous factor or factors, for example, a food poison. Analogous reactions are well-known as illustrated by the production of experimental sarcomas by indol, arsenic, tar, etc. and which have been transmitted by Berkefeld filtrates.

The discovery in recent years of the so-called Coxsackie virus has tended to further confuse the entire poliomyelitis problem. Hoyne[107] (1951), for example, states that the announcement of this discovery "is accompanied by some feeling of dismay. . . . In view of the foregoing announcement it seems that trained investigators have added one more problem to the nebulous conditions enveloping poliomyelitis. One might also be tempted to make the statement that the more we learn about poliomyelitis, the less we know." Hoyne's statement applies obviously to the confusion that has arisen

from exclusive virus studies in poliomyelitis. A Lancet editorial[108] (1951) also indicates the complexity of the problem brought about by the discovery of the Coxsackie virus, as follows: "A crop of new snags is coming along as every week brings new tidings of the Coxsackie viruses."

Many diseases have been considered to be caused by viruses but virus studies constitute only a portion of the investigations intended to determine the cause of the disease. Poliomyelitis investigations, on the other hand, have been confined exclusively to virus studies. Because of this situation and the Public Health Law, those who maintain other opinions, including those concerning poisons as the cause of poliomyelitis, can neither obtain funds from any source for research nor cooperation for investigating their ideas. Reappraisal and investigation of all theories, infectious and non-infectious, are imperative.

To epitomize, the following quotation from a talk on poliomyelitis given by Dr. Ritchie Russell,[97] Department of Neurology, United Oxford Hospitals, Oxford, England (1950) summarizes what has been pointed out in the foregoing: "The time is ripe for a survey of our knowledge of the disease, of the methods of treatment we are accustomed to use and our efforts to advance knowledge of the condition. . . . Surely if the Americans with all their millions of dollars for research on poliomyelitis can do so little, we need not try. . . . I do not intend here to praise good work that has been done, but more to emphasize the gaps in our knowledge of the disease in the hope that others may be encouraged to work on the subject which sorely needs some extra attention. There are at present several different groups of specialists working on the disease, including virologists, infectious disease physicians, orthopedic surgeons, physiotherapists and public health officers. This may be a reasonably adequate state of affairs as far as handling the individual case is concerned; but as a background for advancing knowledge of the disease it is not satisfactory for the members of each of these groups have many other interests, and have neither the time nor always the experience of other aspects of the disease to enable them to fit their piece into the whole picture in such a way as to advance research. . . . Unfortunately, the disease gets worse as public health improves, and measures which are designed simply to avoid infection seem to be singularly ineffective in poliomyelitis."

This statement is reminiscent of one made in 1938 by Dr. Carl C. Dauer,[109] Chief of the Bureau of Preventable Diseases, District of Columbia Health Department, viz: "It seems rather remarkable in spite of all the time and effort spent in poliomyelitis studies during the past 20 years so little information in the epidemiology of the disease has been produced."

The urgent need for cooperation between the clinician and laboratory worker, as well as an unprejudiced attitude toward the poliomyelitis problem, is emphasized by Jungeblut[105] as follows: "The subject is of keen interest to clinicians and laboratory workers alike and progress will be measured by the extent of cooperation between the two. Until the final word has been said it is hoped that, in the future, students of poliomyelitis will preserve an open mind and maintain an open door in their efforts to unravel the mysteries of this baffling disease." Jungeblut indicates the confusion that has been caused by the virus theory when he states: "Actually, the history of this disease has been marked by periods of violent disagreement among scientific workers and by sharp dissension within the ranks of the medical profession."

SUMMARY

1. The poison cause of paralysis has been known for many years. Poliomyelitis, resulting from poisons, has been demonstrated clinically in humans and produced artificially in experimental animals.

2. A relationship between harvest and poliomyelitis has been suggested repeatedly. At such times, corresponding with epidemics of poliomyelitis, some foods of harvest may be poisonous as a result of fertilizers, insect sprays and intrinsic chemical changes.

3. Some so-called virus diseases and virus inclusions may result from poisons.

4. Mistakes have been made in the past whereby diseases considered to be infectious and communicable were later conclusively shown to be neither infectious nor communicable but related to vitamin deficiency or food poisoning.

5. The investigation of the poison cause of poliomyelitis has been obstructed by the Public Health Law which has made this disease legally communicable and infectious. This legal acceptance of the infectious origin of poliomyelitis is, moreover, in a large

degree responsible for exclusive virus studies in determining the cause of this disease.

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